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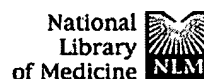
- Nat Med. 1998 Jul;4(7):771

**Cloning and functional analysis of human p51, which structurally :
functionally resembles p53.****Osada M, Ohba M, Kawahara C, Ishioka C, Kanamaru R, Katoh I, Ikawa Y,
Nimura Y, Nakagawara A, Obinata M, Ikawa S.**Department of Cell Biology, Institute of Development, Aging and Cancer, Tohoku
University, Sendai, Japan.

The p53 tumor suppressor gene, which is induced by DNA damage and/or stress stimuli, causes cells to undergo G1-arrest or apoptotic death; thus it plays an essential role in human carcinogenesis. We have searched for p53-related genes by using degenerate PCR, and have identified two cDNA fragments similar to but distinct from p53: one previously reported, p73, and the other new. We cloned two major splicing variants of the latter gene and named these p51A and p51B (a human homologue of Ket). The p51A gene encodes a 448-amino-acid protein with a molecular weight of 50.9 kDa; and p51B, a 641-amino-acid protein with a molecular weight of 71.9 kD. In contrast with the ubiquitous expression of p53, expression of p51 mRNA was found in a limited number of tissues, including skeletal muscle, placenta, mammary gland, prostate, trachea, thymus, salivary gland, uterus, heart and lung. In p53-deficient cells p51A induced growth-suppression and apoptosis, and upregulated p21waf-1 through p53 regulatory elements. Mutations in p51 were found in some human epidermal tumors.

PMID: 9662378 [PubMed - indexed for MEDLINE]

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Services**p63, a p53 homolog at 3q27-29, encodes multiple products with transactivating, death-inducing, and dominant-negative activities.****Yang A, Kaghad M, Wang Y, Gillett E, Fleming MD, Dotsch V, Andrews NC, Caput D, McKeon F.**Department of Cell Biology, Harvard Medical School, Boston, Massachusetts 021
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We describe the cloning of p63, a gene at chromosome 3q27-29 that bears strong homology to the tumor suppressor p53 and to the related gene, p73. p63 was detected in a variety of human and mouse tissues, including proliferating basal cells of epithelial layers in the epidermis, cervix, urothelium, and prostate. Unlike p53, the gene encodes multiple isotypes with remarkably divergent abilities to transactivate reporter genes and induce apoptosis. Importantly, the predominant p63 isotypes in many epithelial tissues lack an acidic N terminus corresponding to the transactivation domain of p53. We demonstrate that these truncated p63 variants can act as dominant-negative agents toward transactivation by p53 and p63, and we suggest the possibility of physiological interactions among members of the p53 family.

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